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Agriculture Development Fund

# FINAL REPORT

## MANAGING LEAD EXPOSURE AND TOXICITY IN COW-CALF HERDS TO MIMIMIZE THE POTENTIAL FOR FOOD RESIDUES AND HERD ECONOMIC LOSSES

**19990255**

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# Managing Lead Exposure and Toxicity in Cow-Calf Herds to Minimize the Potential for Food Residues and Herd Economic Losses

**Project 19990255**

**FINAL REPORT**

Prepared for:

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**Abstract**

Lead is a commonly diagnosed cause of toxicity in cattle. In this study 3 groups of cattle accidentally pastured with discarded lead batteries were intensively studied to determine the extent and severity of exposure. The losses from death due to lead toxicity were substantial in all 3 study herds. Blood samples were taken from all cattle near the time of first diagnosis and then later in 2 of the 3 herds to monitor the change in lead concentrations over time. The asymptomatic lead toxicity observed in these herds and its potential impact on public health has not, however, been previously well documented. In these 3 examples, between 4 and 12% of cattle with no clinical signs had blood lead concentrations considered to be toxic and between 7 and 40% of these asymptomatic animals were in the high-normal range. All cattle exposed to a lead source should be tested prior to sale or slaughter because exposed animals not showing clinical signs may not be suitable for entry into the food chain. The blood lead concentrations were monitored and decreased slowly after the cattle were removed from the lead source possibly due to continued absorption from lead particles in reticulum or rumen.

## Executive Summary

Lead poisoning is the most common toxicity in cattle in western Canada. Lead poisoning is both a food safety issue and an important cause of economic loss in beef and dairy herds. There has, however, been very little research on the field management of lead exposure in beef cattle. In this study, all exposed cattle were tested from 3 herds where group level exposure occurred to old lead batteries. This was unusual in that records from Prairie Diagnostic Services suggest that in almost all diagnostic cases previously submitted to the laboratory only animals with clinical signs were tested. As animals without clinical signs are not regularly being tested, they are likely entering the food chain at some time after lead exposure and have not been considered a potential risk to food safety. In three examples of group exposures studied here, we found that between 4 and 12% of cattle with no clinical signs had toxic blood lead concentrations and between 7 and 40% had blood lead concentrations in the high-normal range suggesting substantial lead exposure. These data suggest that all cattle exposed to a lead source such as discarded batteries should be tested prior to sale and that exposed animals not showing clinical signs may not be suitable for immediate entry into the food chain.

Because of the associated public health considerations, veterinarians need to understand how lead is absorbed, stored, and excreted in cattle to make rational decisions on the management of exposed animals. The oral absorption of lead salts and metallic lead is slow and incomplete. Only 2-10% of ingested lead is absorbed and insoluable lead complexes are excreted in the feces. Once absorbed by the gut, lead is rapidly distributed to soft tissues by the blood, in which most of the lead is carried by red blood cells. Lead is deposited in the kidneys and liver, can cross the placenta and enter the fetus, and can accumulate in the CNS of neonates due to their immature blood-brain barrier. Lead is excreted by active transport into bile, elimination of lead in urine, excretion by lactation, and redistribution of lead into bone.

Seventeen animals with high or toxic blood lead concentrations were tested either 2 or 3 times during the investigation. The changes in blood lead over time were highly variable between animals. Some had a higher blood lead concentration at the second test period than the first suggesting continued exposure or absorption from lead particles remaining in the reticulum or rumen. Because the data were very limited, a simple single compartment model was chosen

to estimate half-life. Additional study with information on the distribution of lead in the rumen, reticulum, blood, liver, bile, kidney, urine, and bone of these animals would be necessary for a more complex and perhaps accurate model of continued absorption and redistribution between various body tissues. The data do suggest, however, that the half-life for blood concentrations associated with ingestion of lead from old batteries may be at least 3 months.

Heifers were identified in the first herd that were pregnant, had no clinical signs, but had blood lead levels sufficiently high to make them potentially unsuitable for sale for human consumption. This small group of heifers was allowed to calve. Because lead can cross the placenta and accumulate in the fetus, the viability of the fetuses was questioned. There were no abortions, one stillbirth, and no calf mortalities from these heifers. Blood samples collected from their calves near the time of birth suggested that their calves would potentially be suitable for human consumption (0.010 to 0.095 ppm).

At parturition, there is a substantial increased calcium requirement, which results in the mobilization of calcium from bone. Increased blood lead levels at parturition may be associated with the mobilization of bone lead with the calcium. Lead is excreted in the milk. Additional blood samples from the calves at weaning after prolonged milk consumption from the exposed heifers would be necessary before a final recommendation could be made on their suitability for slaughter. Unfortunately additional blood samples could not be obtained on either the heifers or their calves. Information on the second season fertility of the heifers was also unavailable in this case. Exposed but healthy heifers and cows may be suitable for retention in the breeding herd; however, further study of long-term fertility and tissue lead distribution in their offspring is necessary before this can be broadly recommended.

**Technical Report**

***Background***

Lead poisoning is the most common toxicity in cattle in western Canada (Blakely, 1984b; Yonge, 1989). Lead poisoning is both a food safety issue and an important cause of economic loss in beef and dairy herds (Blakely, 1984a; Bratton, 1984). There has, however, been very little research on the field management of lead exposure in beef cattle (Stair, 1995). We have very little information on the absorption, distribution, and excretion of lead from field exposures. Traditionally most veterinarians have only requested testing for lead in animals showing clinical signs following lead exposure. When exposure to lead is suspected, clinical signs alone an may not be an adequate screening tool to identify which cattle should be tested before they are sold for human consumption. Blood lead concentration does not correlate well with the presence of clinical signs (Osweiler, 1996). Animals that don't show clinical signs may also have ingested substantial amounts of lead and could have sufficiently high tissue lead levels to be unsuitable for human consumption. When exposed cattle are identified, we don't know how long these animals should be held before they can be safely marketed or if they should ever be marketed for human consumption. We have very little information on what affect lead exposure might have on subsequent fertility and calf viability. Finally, if the calf of an exposed pregnant cow does survive, that calf may have unacceptably high tissue lead levels.

In the fall of 1999, a large group of bred replacement heifers was placed on a pasture containing discarded vehicle batteries in southern Saskatchewan. Many animals died; however, several pregnant heifers with high blood lead levels survived. Data from this group of animals were used to investigate the questions raised above. Two additional herds with similar exposure profiles have also been investigated.

***Objectives***

The objectives of this project were:

1. To examine whether any animals exposed to sources of lead and that do not demonstrate clinical signs of lead toxicity will have elevated blood and tissue concentrations.
2. To determine whether high blood lead concentrations in a group of exposed animals will persist above acceptable levels following ingestion of lead particles from discarded batteries.
3. To measure the effect of lead ingestion on the risk of abortion, stillbirth, calf mortality, and subsequent fertility in a group of heifers that ingested lead particles.
4. To determine if the fetuses from exposed pregnant animals will have high levels of lead at birth.

***Materials and Methods***

All animal data were collected under a protocol approved by the University of Saskatchewan Committee on Animal Care and Supply. Three herds with lead exposure were identified for study.

*Herd #1*

A group of 51 bred heifers, 1 steer, and 8 cows with calves were placed on a rented pasture of native grass at the beginning of October 1999. A bull had been pastured with this group from June 1 through August 1, 1999. Between October 9$^{th}$ and October 24$^{th}$ four heifers were found dead on pasture. The local veterinarian identified two additional sick heifers in the group from the pasture in question. Both heifers were depressed and salivating. The affected heifers had decreased rumen motility, twitching muscles (including their ears), and pasty, foul smelling manure. The temperature and heart rate of both heifers were normal. Based on clinical examination and the history of acute deaths, the local veterinarian tentatively diagnosed lead poisoning. Blood samples from the two sick heifers were collected and submitted for lead analysis. The two sick heifers were subsequently treated with intravenous CaEDTA. The local veterinarian also examined the animal that died on October 24, 1999 and found no significant

lesions. On October 26, 1999, the diagnosis of lead poisoning was confirmed by the veterinary laboratory. Blood lead levels for the two sick heifers were 0.45 and 1.2 ppm.

On October 27, 1999, the cattle were moved from the rented pasture to a corral on the home quarter. A fifth animal died at home in the corral. The two previously treated animals and another heifer were subsequently euthanized for humane reasons by the herd owner. Kidney samples were collected for subsequent analysis. Two more animals had also been treated for signs of lead poisoning.

The rented pasture was examined and the herd owner found two old vehicle batteries. One battery was broken open, with pieces of the lead plates scattered in several places. The second battery was relatively intact. Evidence of fresh manure was found all around the battery pieces and throughout the junk pile. There was also evidence that cattle had been recently grazing in this area.

As of November 01, 1999, 8 animals had died of the 68 that had been grazing on the rented pasture. The owners of the cattle reported that the remaining animals were eating well and no new clinical cases had been identified. On November 1$^{st}$, blood samples were collected from all cattle that had been grazed on the rented pasture and the animals were individually identified, examined for pregnancy, scored for body condition.

*Herd #2*

In late July 2000, a cow and calf were reported to be acting strange. The cow was walking through fences, falling down and not eating. The cow died and kidney samples were sent to the lab for lead analysis. The calf demonstrated classical symptoms of lead poisoning, including blindness, teeth grinding, rumen stasis, anorexia, and dry feces. Blood from this calf was sent to the lab for lead analysis. The calf was treated and eventually recovered from its' symptoms. The kidney from the cow, and the blood from the calf were both diagnostic for lead toxicity.

The source of the lead was identified as the remains of batteries in the pasture at a spot where an old tool shed had burnt down. The area was raked up. More losses occurred and then

topsoil from the area was removed and buried. Twenty-two animals had been on this pasture. Four deaths occurred.

On October 16, 2000, blood samples were collected from twenty-five animals for lead analysis. Twenty-five were sampled because of some uncertainty about which animals had been in that pasture at the time of exposure. Animals with the highest blood lead levels in October were retested on February 23, 2001. Three finished steers were retested again on June 2, 2001.

*Herd #3*

On July 20, 2001, a local veterinarian examined a steer, which was the fourth death that week in the group of 128 steers the owners had been backgrounding since last October. The lead concentration in the liver from this steer was considered to be the toxic range at 8.92 ppm. On July 22, 2001, the local veterinarian treated another steer showing severe depression. It appeared to be improving then died on July 24, apparently of bloat. The pathologist reported cerebrocortical spongiosus with neuronal degeneration. The lead concentration in the liver was 6.35 ppm also indicative of lead poisoning. Five deaths were attributed to lead poisoning with two of the five confirmed with laboratory analysis. Due to drought conditions, the producer had been planning to send all the steers immediately for finishing at a feedlot. On July 27, 2001, ninety animals from the remaining group had blood samples collected for lead analysis. The final thirty-four were sampled on August 8, 2001.

*Laboratory testing*

The blood lead concentrations were determined on a Model 3010A esa Trace Metals Analyzer which uses anode stripping voltametry. The reagent used in this analysis was Metexcahnge M. To determine tissue levels of lead, the organic matter was first digested with nitric acid and then reconstituted to 10 mL with distilled deionized water. An atomic absorption spectrophotometer set at a wavelength of 217 nm was then used to determine tissue lead levels.

The toxicology laboratory defines a normal blood lead concentration as less than 0.1 ppm. Blood lead levels between 0.1 and 0.3 ppm are classified as high-normal and are reflective of exposure to lead. Levels above 0.3 ppm are considered diagnostic for lead toxicity.

*Statistical analysis*

All data were summarized on commercially available software programs. The change in blood lead concentrations over time were examined using a multi-level model to account for multiple observations from individual animals and the correlation of individual animal responses within herds (PROC MIXED) by assigning a random term for animal identification clustered within herd. Blood lead was modeled as a function of time since first diagnosis. A second order term for time since first diagnosis (time squared) was also introduced into the model but did not improve the model fit or affect the association between time and lead concentration and was then dropped.

The half-life for the elimination of lead was estimated for each pair of blood lead concentrations within individual animals using a simple single compartment model for exponential elimination (Buck et al., 1976). The median and interquartile range were then calculated for the distribution of resulting half-lives calculated from the data.

*Records search*

A search of the record system for Prairie Diagnostic Services was used to locate all of the cases of lead poisoning examined by their laboratories from January 1, 1999 through November 31, 1999.

***Results***

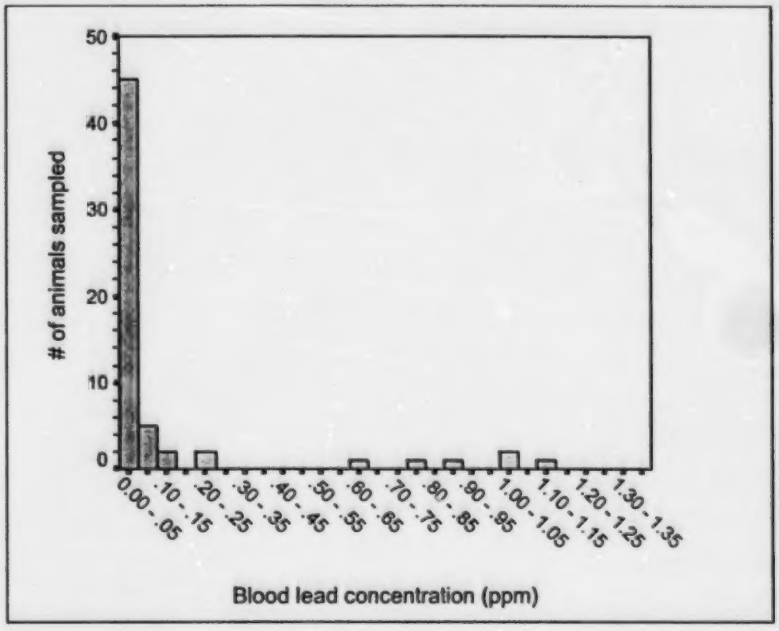
*Herd #1*

Tissue lead for the kidney samples taken from the three heifers euthanized by the herd owner were 18.9, 13.3 and 18.9 ppm.

One calf with previously unrecognized clinical signs was identified during blood sample collection in November. Five of the remaining 60 animals (8.3 %) that were on the contaminated pasture had blood lead levels greater than 0.3 ppm (Figure 1). Surprisingly, three of these six animals (4.2 %), had blood lead levels considered toxic, yet did not show any clinical signs of lead toxicity on the date of the farm visit. All of the animals with toxic blood lead levels were young stock, 5 were heifers and one was a calf. Four of the five heifers with toxic blood lead levels were pregnant. The size and consistency of the uterus for the non-pregnant heifer suggested that she might have aborted.

Four more animals (6.7%) had blood lead levels in the high normal range (0.1 – 0.3 ppm), which indicated significant exposure to lead but not necessarily clinical toxicity. All four of these animals were heifers and were pregnant. All other animals that had been on the rented pasture had blood lead levels within the normal range suggesting no substantial ingestion of lead.

Figure 1. Distribution of blood lead concentrations (ppm) in potentially exposed calves from Herd 1.



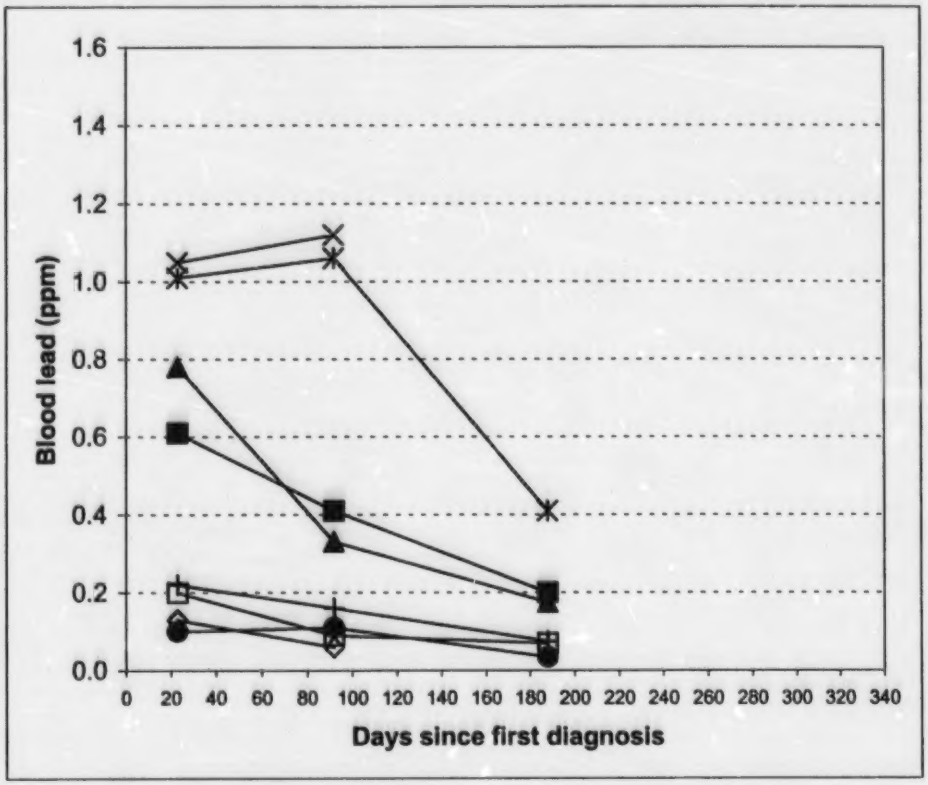

Another heifer died on Dec 01, 1999. This heifer had been treated on the pasture, and had toxic blood lead levels when examined on November 01, 1999. The kidney lead from this heifer was 15.4 ppm. Additional blood samples were collected on January 9, 2000 from all surviving animals except the 1999-born steer calf (Table 1).

Table 1. Blood lead concentrations (ppm) and reproductive outcomes for exposed heifers in Herd 1.

| Study Number | Year born | Pregnancy status as of Nov 1, 1999 | Blood lead (ppm) as of Nov 1st | Interpretation | Blood lead (ppm) as of Jan 9, 2000 | Blood lead (ppm) as of April 14, 2000 | Comments |
|---|---|---|---|---|---|---|---|
| 508 | 1998 | Pregnant | 1.12 | Toxic | *dead* | *dead* | *clinical signs* |
| 511 | 1999 | N/A- calf | 0.89 | Toxic | | | *no sample submitted Jan or April 2000 – clinical sign* |
| 516 | 1998 | Pregnant | 0.61 | Toxic | 0.41 | 0.20 | Calf sample 0.095 ppm |
| 517 | 1998 | Pregnant | 0.78 | Toxic | 0.33 | 0.175 | Calf sample 0.025 ppm |
| 522 | 1998 | Pregnant | 1.05 | Toxic | 1.12 | -- | *no sample submitted April 2000* |
| 524 | 1998 | Open | 1.01 | Toxic | 1.06 | 0.410 | *Open – clinical signs* |
| 528 | 1998 | Pregnant | 0.10 | Exposed | 0.11 | 0.035 | Calf was stillborn – no sample collected |
| 548 | 1998 | Pregnant | 0.20 | Exposed | 0.09 | 0.070 | Calf sample 0.020 ppm |
| 553 | 1998 | Pregnant | 0.13 | Exposed | 0.06 | -- | *Cow killed by lightning* |
| 555 | 1998 | Pregnant | 0.22 | Exposed | 0.16 | 0.075 | Calf sample 0.010 ppm |

There was no significant difference between the November and January blood lead levels for the heifers that were bled on both occasions ($P > 0.05$) (Figure 2). The samples collected in April were, however, significantly lower than the samples collected in November. None of the 8 heifers confirmed pregnant aborted prior to calving and one heifer lost her calf at birth. None of the 7 surviving calves from these heifers died between birth and weaning.

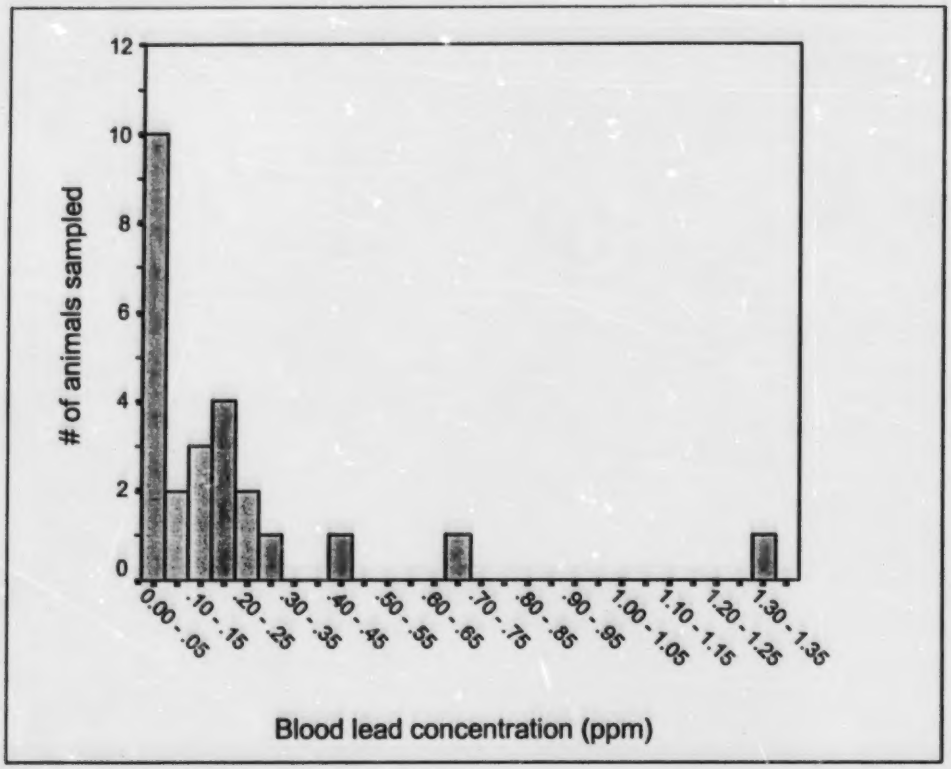
Figure 2. Change in blood lead concentration (ppm) over time for exposed heifers from Herd 1.




*Herd #2*

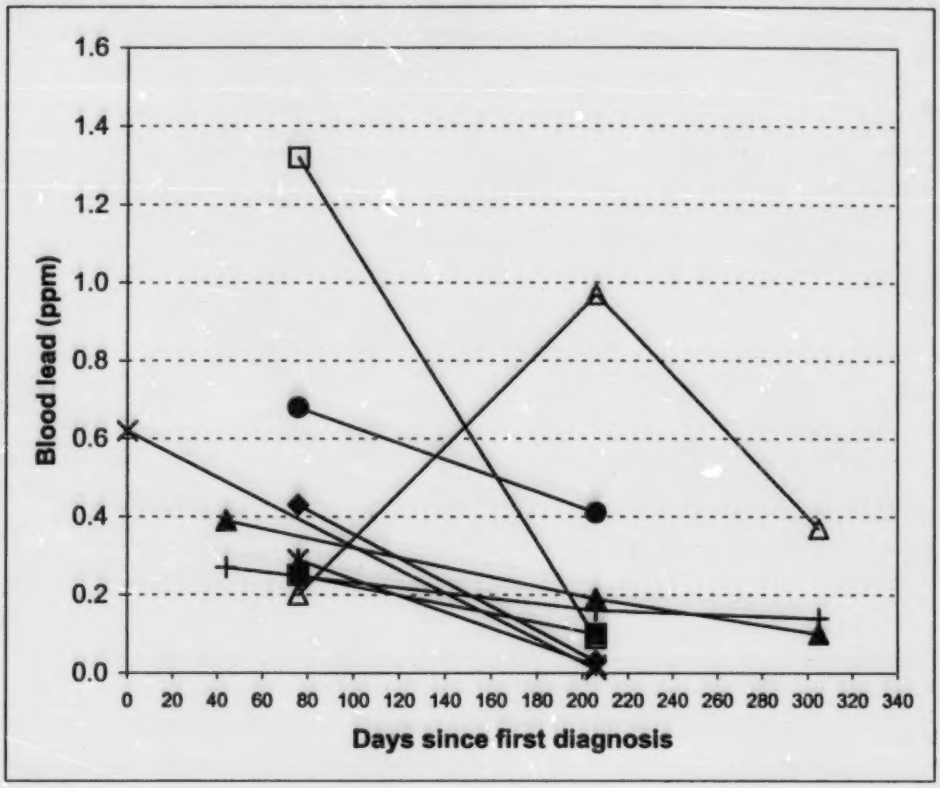
Of the 25 asymptomatic animals that were examined, 3 (12%) were identified in the toxic range, and 10 (40%) were in the high normal range (Figure 3).

Figure 3. Distribution of blood lead concentrations (ppm) in potentially exposed calves from Herd 2.




Ten of these animals were retested one or more times to monitor any change in blood lead concentrations.

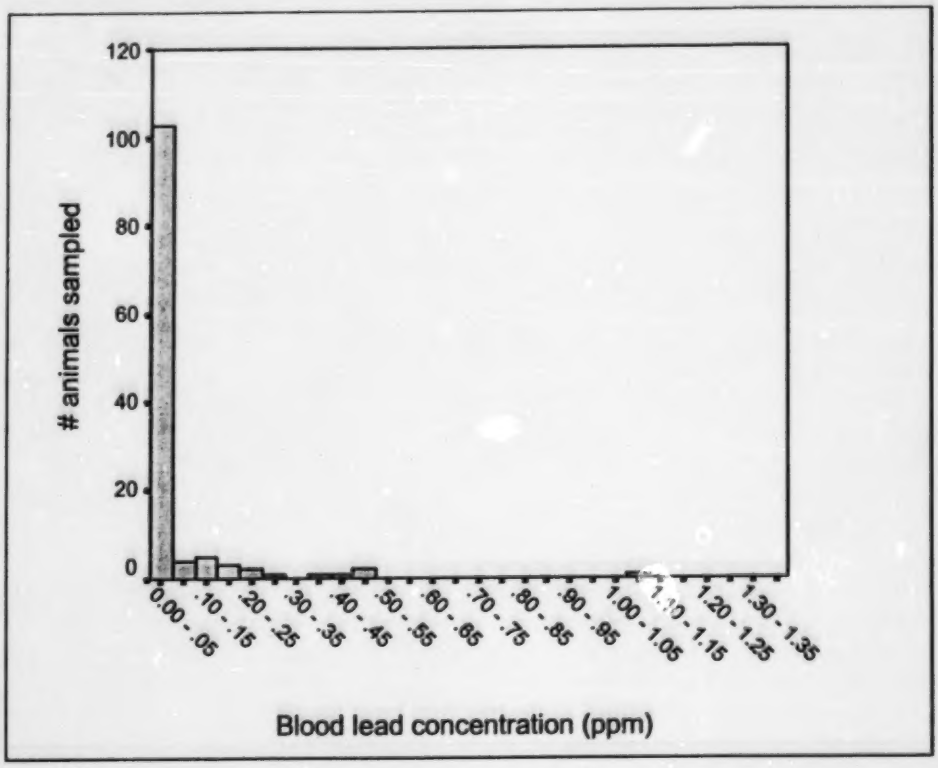
Figure 4. Change in blood lead concentration (ppm) over time for exposed cattle from Herd 2.




*Herd #3*

The surviving exposed steers showed no clinical signs with the possible exception of one animal. This calf had been acting unusually one week previous to the deaths but recovered without treatment. Blood testing identified 5 more animals in the toxic range (4.1%), and 11 animals in the high normal range (8.9%).

Figure 5. Distribution of blood lead concentrations (ppm) in potentially exposed calves from Herd 3.




*Changes in blood lead concentration after exposure*

Individual animals in herd 1 and herd 2 were sampled at 2 or 3 time points to monitor the decrease in blood lead concentration. The mean decrease in blood lead was 0.046 ppm (95% CI, 0.017 to 0.075 ppm) every 30 days for these two herds. Using a single component exponential model the median half-life of lead in the animals retested from herds 1 and 2 was 63 days (interquartile range, 34 to 107 days).

***Conclusions***

The economic losses from death due to lead toxicity were substantial in all three study herds. The clinical signs and occurrence of deaths from battery ingestion are very common and have been well documented (Gudmundson, 1983; Blakely, 1984b; Hoff et al., 1998). The asymptomatic lead toxicity observed in these herds and its potential impact on the safety of beef from exposed herds has not, however, been well documented.

In this study, all exposed cattle were tested from 3 herds where group level exposure occurred to old lead batteries. This was unusual in that records from Prairie Diagnostic Services suggest that in almost all diagnostic cases submitted to the laboratory only animals with clinical signs were tested. As animals without clinical signs are not being tested, they are likely entering the food chain at some time after lead exposure and have not been considered a potential risk to human health. In these 3 group exposures, we found that between 4 and 12% of cattle with no clinical signs had toxic blood lead concentrations and between 7 and 40% had blood lead concentrations in the high-normal range suggesting substantial lead exposure. These data suggest that all cattle exposed to a lead source such as discarded batteries should be tested prior to sale and that exposed animals not showing clinical signs may not be suitable for immediate entry into the food chain.

Because of the associated public health considerations, veterinarians need to understand how lead is absorbed, stored, and excreted in cattle to make rational decisions on the management of exposed animals. The oral absorption of lead salts and metallic lead is slow and incomplete (Osweiler, 1996). Only 2-10% of ingested lead is absorbed and insoluable lead complexes are excreted in the feces. Once absorbed by the gut, lead is rapidly distributed to soft tissues by the blood, in which the majority of the lead is carried by red blood cells. Lead is deposited in the kidneys and liver, can cross the placenta and enter the fetus, and can accumulate in the CNS of neonates due to their immature blood-brain barrier. Lead is excreted by active transport into bile, elimination of lead in urine, excretion by lactation, and redistribution of lead into bone. Bone is the tissue that serves as the long-term storage site for lead, with a half-life of 5 to 30 years. The initial deposition of lead in bone is at areas of active bone growth, such as the

epiphyseal plate. Over time, the lead is deposited in cancellous bone, and can account for 60% of the total body burden.

Seventeen animals with high or toxic blood lead concentrations were tested either 2 or 3 times during the investigation. The changes in blood lead over time were highly variable between animals. Some had a higher blood lead concentration at the second test period than the first suggesting continued exposure or absorption from lead particles remaining in the reticulum or rumen. Because the data were very limited, a simple single compartment model was chosen to estimate half-life. Baars et al. (1992) used this method to calculate a biological half-life of 10.5 days (SE, 1.1 days) for blood concentrations in a large group of cattle exposed to lead contaminated feed. Additional study with information on the distribution of lead in the rumen, reticulum, blood, liver, bile, kidney, urine, and bone of these animals would be necessary for a more accurate model of continued absorption after the final ingestion and elimination. The data do suggest, however, that the half-life for blood concentrations associated with ingestion of lead from old batteries may be at least 3 months.

Heifers were identified in the first herd that were pregnant, had no clinical signs, but had blood lead levels sufficiently high to make them potentially unsuitable for sale for human consumption. This small group of heifers was allowed to calve. Because lead can cross the placenta and accumulate in the fetus, the viability of the fetuses was questioned. In an experiment where ewes were fed lead through gestation, a lambing rate of 18% was recorded compared with 100% in the control group (Sharma and Buck, 1976). Over 27% of the exposed group aborted while there were no abortions in the control group. There were no abortions, one stillbirth, and no calf mortalities. Blood samples collected from their calves near the time of birth suggested that their calves would potentially be suitable for human consumption (0.010 to 0.095 ppm).

At parturition, there is a substantial increased calcium requirement, which results in the mobilization of calcium from bone. Increased blood lead levels at parturition may be associated with the mobilization of bone lead with the calcium (Galey et al., 1990). Lead is excreted in the milk. Additional blood samples from the calves at weaning after prolonged milk consumption from the exposed heifers would be necessary before a final recommendation could be made. Unfortunately additional blood samples could not be obtained. Information on the second season

fertility of the heifers was also unavailable in this case. Exposed but healthy heifers and cows may be suitable for retention in the breeding herd; however, further study of long-term fertility and tissue lead distribution in their offspring is necessary before this can be broadly recommended.

***References***


Baars AJ, Van Beeg H, Visser IJR, et al. Lead Intoxication in Cattle: A Case Report. Food Additives and Contaminants 1992; 9: 357-364.

Blakely BR. A Retrospective Study of Lead Poisoning in Cattle. Vet Hum Toxicol 1984a; 26: 505-507.

Blakely BR. The Incidence and Seasonal Characteristics of Veterinary Toxicosis in Saskatchewan. Can Vet J 1984b; 25: 17-20.

Bratton GR, Zmudski J. Laboratory Diagnosis of Pb Poisoning in Cattle: A Reassessment and Rewiew. Vet Hum Toxicol 1984; 26: 387-392.

Galey FD, Slenning BD, Anderson ML, et al. Lead concentration in blood and milk from periparturient dair heifers seven months after an episode of acute lead toxicosis. J Vet Diagn Invest 1990; 2: 222-226.

Gudmundson, J. Lead Poisoning in Cattle. Agri-Practice 1993; 14(5): 43-47.

Hoff B, Boermands HJ, Baird JD. Retrospective study of toxic metal analyses requested at a veterinary diagnostic toxicology laboratory in Ontario (1990-1995). Can Vet J 1998; 39: 39-43.

Osweiler GD. Toxicology. Philadephia: Williams & Wilkins, 1996: 191-197.

Sharma RM, Buck WB. Effects of chronic lead exposure on pregnant sheep and their progeny. Vet Tox 1976; 18: 186-188.

Stair EL, Kirkpatrick JG, Whitenack DL. Lead arsenate poisoning in a herd of beef cattle. J Am Vet Med Assoc 1995; 207: 341-343.

Yonge KS, Morden BB. Bovine lead poisoning in Alberta: A management disease. Can Vet J 1989; 30: 42.45.

***Project activities, achievements, papers, presentations, etc. (To date)***

Results of this research will presented to veterinary organizations potentially including the Saskatchewan Veterinary Medical Association and the Western Canadian Association of Bovine Practitioners. Results will also be communicated to interested producer groups at forums potentially including the Saskatchewan Beef Symposium. Results will also be published in lay and technical journals (example, Canadian Veterinary Journal) in addition to the final report requested by Saskatchewan Agriculture and Food.

***No project funds were used for "Personnel" expenses or for the purchase of equipment.***

***There are no applicable Project Developed Materials or Project Photos for inclusion with the report.***

***Acknowledgement***

No results have been presented to date. When the results are finally presented, Saskatchewan Department of Agriculture and Food will be acknowledged for providing partial funding for the project.

***Final Expense Statement***

To be provided by University of Saskatchewan Office of Research Services.